ECHOCARDIOGRAPHIC ASSESSMENT OF LEFT VENTRICULAR ANATOMY AND FUNCTIONS IN PATIENT OF SYSTEMIC HYPERTENSION

THESIS FOR

DOCTOR OF MEDICINE

(INTERNAL MEDICINE)

BUNDELKHAND UNIVERSITY
JHANSI (U.P.)

2003 **PRASHANT KUMAR**

Dedicated

To

Respected

Teachers, Parents,

Friends & my Supportive

Family members

ACKNOWLEDGEMENT

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Date: 7 / 7/ 2003

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Prashant Kumar.

CERTIFICATE

This is to certify that the work entitled ***"Echocardiographic assessment of left ventricular anatomy and functions in patient of systemic hypertension"*** which is being submitted as a thesis for M.D. (Medicine) Examination 2003 of Bundelkhand University, Jhansi, has been carried out by ***Dr. Prashant Kumar*** in the Department of Medicine, M.L.B. Medical College, Jhansi.

This method described was undertaken by the candidate himself and the observations recorded have been periodically checked up. He has put in the necessary stay in the Department as per University regulations, and has fulfilled the conditions required for the submission of thesis according to University regulations.

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CONTENTS

Introduction

INTRODUCTION

Systemic hypertension is now known to be common enough, subtle enough and lethal enough to impress the cardiologist more poignantly than it has traditionally been. Through easily detectable as well as easily treatable, it is notorious for worldwide morbidity and mortality. The reason for this is attributed to its inherent propensity to induce vascular damage, leading to cardiovascular, cerebral, renal and ophthalmic complication. In most countries as many as 15-20 % of adult population are found at screening to have a raised Blood Pressure, about 2/3rd of them have mild hypertension (W.H.O. Bulletin 1993).

As the public and medical profession has become aware of the overall 8 bad consequences of even mild hypertension, enthusiasm for its early recognition and aggressive treatment has continued to mount. As reducing Blood Pressure has been clearly shown to decrease morbidity and mortality from cerebrovascular stroke, accelerated coronary events/congestive cardiac failure and renal failure (Macmonam et al 1990).

Various studies confirm that cardiovascular events related to hypertension comprise its major complications. In untreated cases, fifty percent of hypertensive patients die of cardiac problem, thirty three percent of stroke and ten to fifteen percent of renal failure (Eugene Braunwald, 2001).

The cardiovascular complications of hypertension can be divided into two group : Coronary heart disease is said to exist, if there is left ventricular hypertrophy (LVH) and / or left ventricular dysfunction plus elevated blood pressure.

Asymptomatic patients with abnormal myocardial function due to hypertension need care at an early stage to prevent or retard development of left ventricular failure. This has focussed attention to the need of a technique that could provide information regarding the cardiovascular functional and anatomical status, conveniently.

Noninvasive investigatory procedures that could provide an assessment of left ventricular status are : Chest radiography, Electrocardiography, Radionuclide ventriculo--graphy and echocardiography. X-ray detection of L.V.H. is fraught with the problems of defining which portions of the cardiac silhouette belong specifically to left ventricle. Although there are probable and definite criteria for diagnosis of L.V.H. by E.C.G, but it is in arrhythmias and blocks that E.C.G often gives a clear and irrefutable answer to the knowledgeable interpreter, whereas with myocardial disease, it lacks specificity. Epidemiological studies of hypertension using E.C.G reported a prevalence of 2.1% of L.V.H. in Framinghom cohort. Another using echocardiography which is more sensitive in detecting left ventricular hypertrophy reported a prevalence of 16% (Levy D. et al 1988). Echo is 10 times more sensitive than E.C.G. in documenting L.V.H. (Malloy T.J. et al 1992). Echo Doppler study is modality of choice to document cardiac involvement in hypertension (Mc farland TM et at 1998). Besides, X-ray and E.C.G. are much

less sensitive than echocardiography in this respect, as evidenced by the study carried out, by Savage D.D. et al (1979), showing only 10% 0f hypertensive L.V.H. cases proved by echocardiography, could be detected on X-ray screening and E.C.G.

Radionuclide ventriculography only, like echocardiogra--phy, though can give a functional assessment of left ventricle yet its biggest limitation is its scarce availability even at the highest centres. Besides it is better suited to volume estimates. Its cost effectiveness compared with echocardiography puts it quite back in the scene of specialized investigations.

Thence, echocardiography has emerged as a promising modality. In contrast to radionuclide ventriculogra - - phy, this technique has evolved from a laboratory curiosity, being undertaken in a few widely scattered centres, to the community hospital and even to the individual chamber of cardiologist. This provides valuable information both about any structural as well as functional abnormality of the myocardium at quite an early stage of onset of disease. The non-invasive and harmless nature, easy availability, relative ease of performance, the reliability and reproducibility of the resulting information and the sensitivity and specificity may make it the investigation of choice to detect myocardial abnormality in hypertensive patients.

Thus echocardiography has been a major breakthrough in this respect, and since then many studies have been undertaken to assess the cardiovascular status in hypertensive patients, and to find out its predictor value as

prognostic significance. In view of the increasing importance of myocardial dysfunction in hypertensive individuals, the present study has been undertaken with the aim to detect left ventricular diastolic and systolic function by means of echocardiography, in patients of systemic hypertension.

Review Of Literature

REVIEW OF LITERATURE

History of hypertension in the modern era dates back to Stephen Hales in 1873 who first ventured to investigate blood pressure by the use of saline manometer. Today, though blood pressure measurement is a simple bedside procedure, the criteria for the diagnosis of hypertension has been arrived at after much controversy.

Life insurance actuaries (1959), identified mortality increments in policy holders with what many clinicians considered trivial elevation of blood pressure. Framingham heart study (1968), showed discrete risks for isolated systolic and diastolic elevation. 'HANES' survey of United States (1977) selected a blood pressure of 160/95 mm Hg or more to define hypertension. Perloff & Sokolow (1978) documented the value of 24 hours blood pressure surveillance in interpreting the need for medication, the response to medication and the inherent variability of hypertension measured under a variety of circumstances.

Fowler et al (1980), made the criterion to 95 mm Hg in patients over age 30 years & applied thus standard to recumbent or standing measurements.

Current standards for defining & diagnosing hypertension rests on the Blood Pressure levels which confers an increased risk of developing a morbid cardiovascular event and/or will clearly benefit for medical therapy. In adults, according to the *VIth report of Joint*

National Committee for prevention, detection, evaluation and treatment of high blood pressure the following values are now considered:-

Diastolic blood pressure:-

below 80 mm Hg	Optimal
less than 85 mm Hg	Normal
85 mm Hg – 89 mm Hg	High normal
90 mm Hg –99 mm Hg	Hypertension stage I
100 mm Hg –109 mm Hg	Hypertension stage II
110 mm Hg or above	Hypertension stage III

Systolic blood pressure:-

below 120 mm Hg	Optimal
less than 130 mm Hg	Normal
130 –139 mm Hg	High normal
140 mm Hg – 159 mm Hg	Hypertension stage I
160 mm Hg –179 mm Hg	Hypertension stage II
greater than180 mm Hg	Hypertension stage III

These levels should be persistent and blood pressure should be measured on two separate occasions under the proposed near ideal conditions before labeling a patient as hypertensive. Higher values of systolic or diastolic pressure will be taken for staging.

Normal Cardiac Performance and its Control:

The myocardial function is divided into two cardiac cycles:-

Diastole:– A period of relaxation followed by Systole – a period of contraction.

Diastole:– This is further divided into following phases:-

- ❖ Isovolumic relaxation
- ❖ Rapid inflow
- ❖ Diastasis
- ❖ Atrial systole

Systole:– Similarly this has been divided into:-

- ❖ Isovolumic contraction
- ❖ Period of ejection
- ❖ Protodiastole

The extent of shortening of mammalian heart muscle and therefore the stroke volume of the intact ventricle are in final analysis determined by three influences (Guyton A.C. et al).

1. The length of the muscle at the onset of contraction, i.e. the preload.
2. The inotropic state of the muscle.
3. The tension that the muscle is called upon to develop during contraction i.e. the afterload.

Ventricular Afterload

The afterload on the intact heart may be defined as the tension or stress developed in the wall of the ventricle during ejection. Therefore, the afterload on the ventricular muscle

fibres is also dependent on the level of aortic pressure as well as on the volume and thickness of the ventricular cavity, since Laplace's Law indicates that: –

$$\text{Myocard.Fibre Tn} = \frac{\text{Vent.Rad. x Intra cavity Vent. Press.}}{\text{Wall thickness}}$$

Thus at the same level of aortic pressure, the afterload faced by a dilated left ventricle is higher than that encountered by a ventricle of normal size. (Harrison's principles of internal medicine, 15th ed. Vol. I; pp.1315).

Diastolic properties of the Left ventricle

Left ventricle pressure and volume during diastole, reflect the interaction of ventricular elastic, viscous and inertial properties, and the completeness of myocardial relaxation (Grossman et al). In their study they summarized the factors determining left ventricular diastolic properties as follows:-

Principal factor determining left ventricular diastolic properties

(A) Properties intrinsic to the ventricular chamber

1. Completeness of ventricular relaxation.
2. Passive elastic properties of ventricular chamber (stiffness or compliance).
    (a) Thickness of ventricular walls.
    (b) Composition of ventricular wall (muscle, scar amyloid, calcium, iron.
3. Viscous properties.

4. Diastolic suction (elastic recoil), inertial properties.
5. Influence of contractile state.
6. Influence of other factors (e.g. myocardial ischemia, temperature, osmolality).

(B) properties extrinsic to the ventricle

1. Pericardial properties.
2. Atrial contraction (presence, strength).
3. Increased diastolic inflow (shunts, high output states, valvular insufficiency).
4. Blood volume.
5. Overload of the right ventricle.

Recent studies indicate that for cardiac muscle, relaxation is not a passive, but a complex energy dependent process (Langer et al).

Left ventricular relaxation may be impaired in patients with congestive heart failure, without systolic dysfunction (Litwin JE.et.al 1993).

Diastolic Dysfunction precedes systolic dysfunction in hypertensive patients

Earliest function cardiac changes in hypertension are in left ventricular diastolic function, with prolongation and in coordination of isovolumic relaxation. (Braunwald 2001 page 949, Dibello.V.et al 1999).

Topol et al (1985) found elderly patients with hypertension having abnormal diastolic function, but excessive LV emptying.

Similarly, Pearson et al (1987) found only LV diastolic abnormalities in hypertensive patients with LVH in the form of reduced Doppler determined peak filling rates.

Mechanism of L.V.H in systemic hypertension

Pathogenesis of L.V.H involve a number of variables other then the pressure load, one of which is haemodynamic volume load. Devereux and colleagues (1992) found a close correlation between left ventricular stroke volume and left ventricular mass, with diastolic then with systolic blood pressure. Other determinants are obesity (Gottdiener et al 1997), levels of sympathetic nervous system and renin angiotensin activity and whole blood viscosity (Braunwald et al 1994). The correlation is much closer between L.V.H. and pressure readings taken during the stress of work by ambulatory monitoring, than between L.V.H. and casual pressure reading (Gottdiener et al 1994). By echocardiography, left ventricular mass is shown to progressively increase with increase in blood pressure (Kahan T. et al 1998). Left ventricular mass is greater in those whose pressure does not fall during sleep because of a more persistent pressure load (Deveurex et al 1992).

Different pattern of hypertrophy may evolve often starting with asymmetrical left ventricular remodeling from isolated septal thickness, which has been noted in 22% of untreated hypertensives with normal total left ventricular mass (Verdecchia et al. 1994).

L.V.H an independent risk factor

Studies have shown that L.V.H. has emerged as a powerful independent prognostic factor (Casale PN et al 1986, Deverux RB et al 1989, Lavy D.et al 1990, Aurigemma GP et al 1995, Kannel WB et al 1991) for cardiac mortality over and above the extent of coronary artery disease (Kahan T et al 1998). In addition risk of ventricular arrhythmias is increased at least two fold in presence of L.V.H. (Ichkhan et al 1997). Increased incidence of sudden cardiac death in L.V.H. is thought to be arrhythmogeneic in origin (Mclenachan et al 1987).

Treatment with all antihypertensive drugs except those that further activate sympathetic nervous activity, e.g.,direct vasodilators such as hydralzine when used alone has been shown to cause L.V.H. regression (Ofili EO et al 1998) with regression, left ventricular function usually improves and cardiovascular morbidity decreases (Verdecchia et al 1998).

Relationship between L.V.H. and L.V. dysfunction in patients of systemic hypertension

Dreslinski et al (1981) studied 10 normal subjects, 11 hypertensive patients without echocardiographic evidence of L.V.H. and 10 patients of hypertension with L.V.H. They observed that progressive decrease in diastolic function in the 3 groups respectively.

Systolic dysfunction, however does not correlate well with L.V.H. in hypertensive cases. Toshima et al reported a normal echocardiographic ejection fraction in 11 patients with concentric L.V.H caused by hypertension.

LV Dysfunction in hypertensive patients with CHF

Heart failure is an abnormality of cardiac function responsible for the inability of the heart to pump blood at a rate commensurate with the requirements of the metabolizing tissues and/ or can do only from an abnormally elevated filling pressure.

Abnormalities during systole and/or diastole may be present in heart failure (Vasan RS et al 1996). Blacks are more prone, to progression to heart failure and death than whites. In so called systolic heart failure i.e. classic heart failure, an impaired inotropic state causes weakened systolic contraction which leads, ultimately to a reduction in stroke volume, inadequate ventricular emptying, cardiac dilatation, and often elevation of ventricular diastolic pressure. Traditionally, CHF consequent to hypertension and was considered to have only systolic dysfunction. In diastolic heart failure the principal abnormality involves impaired relaxation of the ventricle and a normal diastolic volume. Failure of relaxation can be caused by a stiffened thickened ventricle as in hypertension. So hypertension is common cause of diastolic dysfunction and diastolic heart failure (Dougherty AL et al 1984). Recognition of this fact has a lot of clinical relevance in terms of treatment.

Dougherty et al (1984) studied 184 patients with CHF among which 64% had reduced EF and 36% turned out to have normal EF. On further analysis 65% cases of those normal EF turned out to be cases of hypertension. So they concluded that among patients with clinical heart failure

normal global systolic function is common, and that the pulmonary congestive symptoms are associated with abnormal LV compliance. Also they did not find any correlation between severity of symptoms and EF. Another observation made by them was the finding, that internal ventricular dimensions in 13 out of 16 patients with a normal EF were normal by quantitative echocardiography, which is more direct measure of chamber size. Thus in their study patients of hypertension had isolated diastolic, isolated systolic and combined dysfunction as well. Most episodes of C.H.F. in hypertensive patients are associated with dilated cardiomyopathy and a reduced ejection fraction, however, about 40% episodes of C.H.F. are associated with preserved L.V. systolic function, but with diastolic dysfunction induced by L.V.H., fibrosis and ischemia and increased afterload (Bonow & Udelson 1992).

Echocardiographic parameters of LV function

Safar et al (1990), found LVH in hypertensive patients by the increase in IVS and LVPW, which was statistically significant ($p < 0.001$ and $p < 0.01$ respectively).

LVIDd and LVIDs were not different from normal diameters in subjects with sustained hypertension except for LVIDs which was significantly increased. The relative reduction in diameter (LVIDs – LVIDs/LVIDd) was equal in normotensive subjects and patients with borderline hypertension (0.33 ± 0.08 VS 0.30 ± 0.06). It showed some decrease in patients with sustained hypertension (0.26 ± 0.09) but the difference was not statistically significant.

Savage et al (1989) found increased IVSd in 50%, increased LVPW (61%) and increased LV mass in 51% of hypertensives, and this increase was statistically significant. LVEF was increased in 15% and EF slope decreased in 6%. Mean values of LVIDd and LVIDs, left atrial and aortic root dimension and LVEF for the hypertensive subjects were not significantly different from values in normal subjects.

There was no significant correlation (positive or negative between ventricular septal or LV free wall thickness and EF. Although only a small number of hypertensive subjects had a mitral valve EF slope below the 95% prediction interval, the mean EF slope of hypertensive subjects was significantly lower than that of normal subjects ($p < 0.001$).

Nine hypertensive subjects had ventricular septal thickening that was disproportionate to the LV free wall thickness (i.e. septal free wall ratio $\geq$ 1.3). Their measured septal thickness ranged from 15 -27 mm with septal free wall ratios from 1.3 – 1.9. None of the nine had systolic anterior motion of the anterior leaflet of the mitral valve. However, one subject who had concentric LV wall thickening did have systolic anterior motion of the anterior mitral leaflet.

Hanrath et al (1992), observed that mean LVIDd and LVIDs in the patients of chronic overload was 48.5 ± 7.9 and 26.9 ± 6.1 mm respectively. LVIDs was significantly reduced in comparison with control group ($p < 0.05$). The sum of IVSd and LVPWd (29.1 ± 48 mm) was greater than that in normal subjects ($p < 0.001$).

Assessment of LV diastolic dysfunction by echocardiography

Good pulse wave spectral Doppler transmitral flow pattern recorded at the tips of the mitral leaflets in apical 4 Chamber view show following parameters as suggestive of LV diastolic dysfunction (Oh J.K. et al 1994).

- ❖ Low E velocity and high A velocity.
- ❖ Reversed E/A ratio (<1).
- ❖ Prolonged 'E' deceleration time (DT) (>240 m sec).
- ❖ Prolonged isovolumetric relaxation time (IVRT) from aortic closure to MV opening (<110 m sec).

Normal values of E = 0.85 ± 16 m sec

A = 0.56 ± 0.13 m sec

E/A ratio = 1.6

Assessment of left ventricular hypertrophy or enlargement

In X-ray cardiothoracic ratio is normally well below 50% in PA view but in AP films normal value can be accessed as 55%. In infants the normal valve can be 55%. As the left ventricle enlarges, there is usually an increase in cardiothoracic ratio and curvature of lower left heart border takes on large radius ventricle enlarges towards lateral wall of thorax in a downward direction displacing apex laterally and inferiorly. In lateral view we calculate distance from posterior aspect of inferior venacava to the posterior border of heart horizontally at the level 2 cm above intersection of the diaphragm and the inferior venacava. This is known as

Hofman sign. A distance of greater than 1.8 cm indicate left ventricular enlargement. Such measurements can be helpful but great reliance cannot be placed on them as individual anatomical variation can cause discrepancies (David Sutton 2003).

In ECG for LVH detection Ramhilt and Ester point score system was used. Criteria are as follows:

		Points
1. R or S in limb lead	: ≥ 20 mm	3
SV_1 or SV_2	: ≥ 30 mm	
RV_s or RV_6	: ≥ 30 mm	
2. Intrinscoid deflection in V_5 or V_6 0.05 sec or more	:	1
3. Left axis deviation	: [30^0 or more]	2
4. QRS interval 0.09 sec or more	:	1
5. Left atrial abnormality/enlargement	:	3
6. ST – T changes - without digitalis		3
- with digitalis		1

LVH is present if the total score is more than 5 points and probably present if score is 4 points.

In Echocardiography LV mass was calculated by using formula given by Devereux and Reichek 1977.

$$LVM = 1.04\ [(IVST + LVID + PWT)^3 - LVID^3] - 13.6$$

LV mass index is LV mass per square meter body surface area. LV mass could also be calculated from 2D echo tracing of parasternal short axis showing LV at the

papillary muscle level, showing good endocardial definition by Area Length method. (Schiller N et al 1989). The upper limits of IVST, PWT and LV mass index (gm / m^2) are shown to be 1.1 cm, 1.1 cm and 122 gm/m^2 BSA for Indian men and 0.9 cm, 0.9 cm and 110 gm/m^2 BSA for Indian women (Trivedi et al 1991). In a study by Ghanem Wisam MA et al 2000 LVH was defined by echocardiography as LV mass index > 134 gm/m^2 in men and >110 gm/m^2 in women.

Aims

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Objectives

Aims and Objectives

Echocardiographic assessment of left ventricular anatomy and function in patient of systemic hypertension and comparing the results with normal values of that age and sex.

Material

&

Methods

MATERIAL AND METHODS

Selection of Cases

In the present study, thirty hypertensive patients of both sexes and age ranging between 31 to 68 years, were selected, who either attended the out patient department or were admitted to the indoor wards of M.L.B. Medical College Hospital.

Fifteen control cases were also studied to standardize the various echocardiographic parameters of left ventricular function. These cases were selected from healthy, normotensive attendants of patients.

Criteria for Selection

Study group – 30 cases of hypertension

All patients of systemic hypertension whose diastolic blood pressure was persistently over 90 mm Hg, were selected for the study.

But hypertensive patients with coexistent ischemic heart disease, congenital and acquired heart disease, corpulmonale, pregnancy with or without toxemia, hyperdynamic circulatory states, chronic renal failure and cardiomyopathies were excluded, as these on their own accord, are likely to alter the function of the heart.

Normal Healthy Controls

Fifteen normal healthy individuals of both sexes were studied after careful screening to exclude hypertension or

any other ailment by proper history, clinical examination and investigation.

Methods

The study was done along following lines after informed consent was taken:-

Clinical history and examination – All cases were subjected to detailed clinical history and through physical examination

Investigations – All cases taken up in this study were subjected to following investigations –

(a).Routine

Complete haemogram

Complete urine analysis

Blood sugar – fasting and post prandial

Serum creatinine, Blood urea

Serum Na_+ / K_+

(b).Specific

X-ray chest

E.C.G.

(c).Special

Echocardiography

Chest X - ray

The cases under study were radiologically evaluated for evidence of any cardiac involvement. For this purpose posterioanterior and lateral views of chest were taken. The structural cardiac alteration consequent upon hypertension, which could be detected by X-ray and taken into consideration was left ventricular enlargement.

E.C.G.

A 12 lead electrocardiogram was recorded in all the patients and L.V.H. was considered to exist if, either the probable or the definite criteria of Este's scoring method for LVH was satisfied.

Echocardiography M- Mode, 2 dimensional & Doppler

Cross sectional echocardiography was performed at M.L.B. Medical College Hospital, Medicine department, using Hewlett Packard Ultrasound imaging system, using 3.5 / 2.5 megahertz phase array transducer.

Echocardiography was performed from right side of the patients. To maintain airless contact between transducer and skin, a liquid coupling medium (glycol based or water based) was applied over area of the skin to be scanned.

Patients were examined in supine and left lateral positions and the heart was visualized with multiple transducer positions using all 3 basic echocardiographic windows namely:-

1. Para sternal
2. Apical
3. Subcostal

(a). Parasternal views

1. Parasternal long axis views

It is parallel to plane extending from the right shoulder to left hip. This views allows 2–D examination of the size and shape of left ventricle, right ventricular outflow tract, left atrium and aortic root, as well as examination of mitral and aortic valve.

The transducer is then moved medially to image right ventricle tricuspid valve and right atrium.

2. Parasternal short axis view:

This is obtained by rotating transducer through 90^0 clockwise, from long axis of the heart, parallel to the plane extending from the left shoulder towards right hip.

Examination is done with transducer in 3^{rd} and 2^{nd} interspace pointing directly posteriorly to visualize papillary muscles. Then the transducer is tilted slightly cephalad, to image size of left ventricle during systole and diastole as well as fish mouth appearance of mitral valve.

(b). Apical view

1. Apical four chamber view

This was taken by turning patients to a steeper left lateral position. This view display all 4 cardiac chambers, permits evaluation of ventricular and atrial septa and anterior & posterior leaflets of mitral valve.

2. Five chamber (including aortic outflow)

Beam is angled more anteriorly towards chest wall, 5^{th} chamber is not a chamber but is the aortic valve and ascending aorta.

3. Apical Long Axis view

This is obtained by rotating the transducer 90^0 counter clockwise. This view allows visualization of the ventricular septum and is particularly useful for Doppler examination and to show different segment of left ventricle.

(c). Subcostal view

It is taken with patient lying supine with knees flexed. Transducer is placed in upper epigastrium, pressed firmly back and directed towards patient's head with little posterior and leftwards tilt. This view provides diagnostic yield in patients with chronic pulmonary disease and hyperinflation of lungs and low diaphragm usually precludes examination of the precordial and apical windows. The subcostal four chamber view is particularly helpful in examining the inter atrial and interventricular septum.

The dimensions of the left ventricle and thickness of both the interventricular septum and posterior wall were made at the level of the chordae tendinae in either the parasternal long axis or short axis views.

The left ventricular echocardiographic measurements were made from the endocardial echo of the posterior wall to the endocardial echo of the left side of the interventricular septum. Measurements at end-diastole (LVIDd) were made. The parameters considered for diastolic function were –EF slope, E, A, E/A, for systolic function-LVIDd, LVIDs, EF (ejection fraction). Statistical evaluation of the results were done.

Observations

OBSERVATIONS

The present study was undertaken in 30 hypertensive patients of different age groups, severity and duration of hypertension to assess LV systolic and diastolic functions by means of echocardiography. The study included 15 normal healthy controls to compare with the hypertensive patients. The findings are as follows :

Table.I Showing echocardiographic findings in normal subjects

S. No.	Parameter	Range	Mean ± SD
1.	Left ventricular internal dimension at end diastole LVIDd (cm).	4.2-5.4	4.59 ± 0.39
2.	Left ventricular internal dimension at end systole LVIDs (cm)	2.2-4.0	3.03 ± 0.57
3.	Left ventricular posterior wall thickness at end diastole LVPWD (cm)	0.7-1.2	0.93 ± 0.17
4.	Left ventricular posterior wall thickness at end systole LVPWs (cm)	1.3-1.8	1.56 ± 0.17
5.	Inter ventricular septal thickness at end diastole IVSd (cm)	0.7-1.2	0.93 ± 0.16

6.	Inter ventricular septal thickness at end systole IVSs (cm)	1.1-1.8	1.57 ± 0.16
7.	E F slope	70-120	1.2 ±15.3
8.	Peak flow velocity in early LV filling (E) (cm/s).	80-90	83.3 ± 4.5
9.	Peak flow velocity during atrial Contraction (A)(cm/s).	47-61	52.8 ± 3.9
10.	Ejection fraction of left Ventricle LVEF	65-76%	70.2 ± 3.9
11.	L.V. mass index (g/m^2).	81-98	95.9 ± 9.78

Table. II Age and Sex distribution of control cases

S.No	Age group(in year)	Male	female	Total	Percentage
1.	31-40	2	2	4	26.66
2.	41-50	3	-	3	20.00
3 .	51-60	1	3	4	26.66
4.	61-70	2	2	4	26.66
	TOTAL	8	7	15	100

Range = 31 – 68 years.

Mean ± S.D. = 50.53 ± 11.8

Result

Average age of control cases were found to be 50.53 ± 11.8 (range 31 – 68 years).

Table.III Age and Sex distribution of hypertensive cases

S.No.	Age group(yrs)	Male	Female	Total	Percentage
1.	31 – 40	5	1	6	20
2.	41- 50	7	3	10	33.33
3.	51 – 60	7	3	10	33.33
4.	61 - 70	1	3	4	13.33
	TOTAL	20	10	30	100

Range 31 – 68 years

Mean 49.3 ± 9.8

Result

1. Average age of hypertensive subject is 49.3 ± 9. 8 (range 31 –68 years).

2. Male – Female ratio is 2:1.

Maximum number of cases fall between 41 – 60 years being equally divided in 5^{th} and 6^{th} decades.

Table.IV Prevalence of left ventricular dysfunction in hypertensive patients with or without congestive heart failure (CHF)

S.No	Dysfunction	No. of cases *n=30*	Percentage
1.	Only diastolic dysfunction	19	63.3
2.	Only systolic dysfunction	1	3.3
3.	Both dysfunction	3	10
4.	No dysfunction	7	23.3
5.	Total diastolic dysfunction	22	73.3
6.	Total systolic dysfunction	4	13.3

Result

LV dysfunction was present in 76.6% of hypertensive patients (23 cases out of 30). Rest had no dysfunction. Among (23) case of L.V. dysfunction 95.65% had diastolic dysfunction & 17.39% had systolic dysfunction. 13.04% had both dysfunction & 82.6% had only diastolic dysfunction of the 23 cases, and those with systolic dysfunction only was 4.34%.

Table.V Incidence of LV dysfunction in hypertensive patients with clinically diagnosed CHF

S.No.	Dysfunction	No. of patients *n* =3	Percentage
1.	Only diastolic	1	33.3
2.	Only systolic	1	33.3
3.	Both	1	33.3

Result

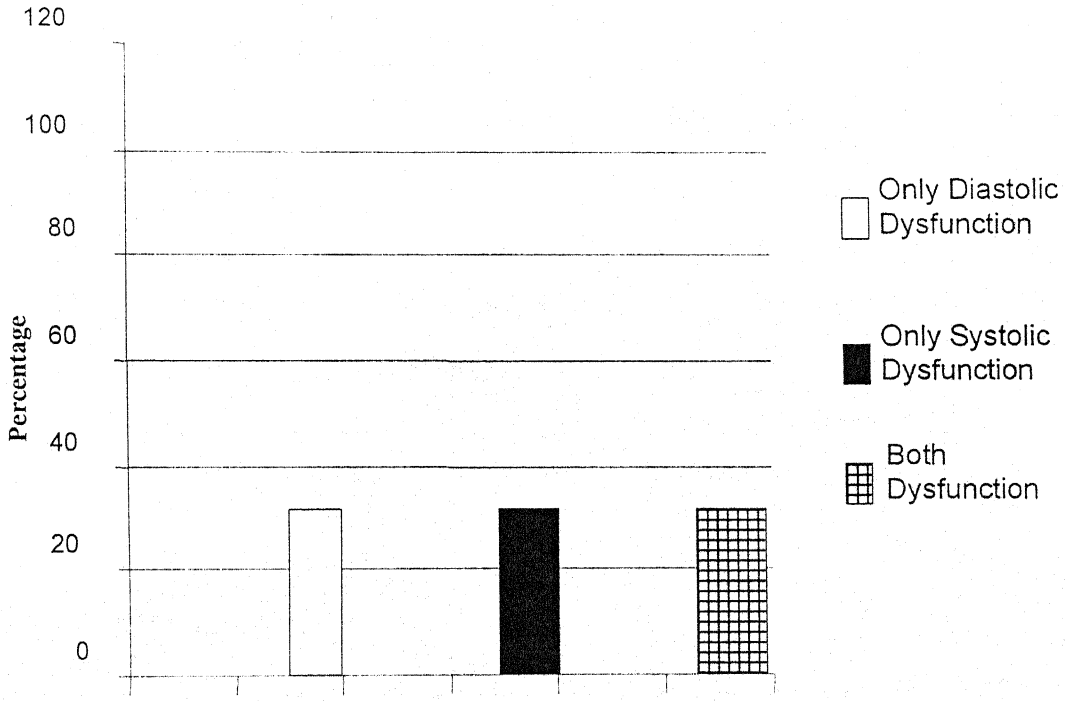
Of 30 cases of hypertension 10% patients i.e. 3 of them presented with CHF. Among them LV diastolic dysfunction, systolic dysfunction & both dysfunction was equally prevalent i.e. 33.3%.

Table.VI Comparison of echocardiographic parameters between normotensive & hypertensive subjects

S. No.	Parameter	Normotensive *(n=15)* Mean ± SD	Hypertensive *(n=30)* Mean ± SD	t	p	Inference
1.	LVIDd	4.59 ± 0.39	4.8 ± 0.52	1.35	>.05	Insignificant
2.	LVIDs	3.03 ±0.57	3.4 ± 0.67	1.84	>.05	Insignificant
3.	LVPWd	0.93 ± 0.17	1.3 ± 0.27	4.87	<.001	Highly Signi
4.	LVPWs	1.56 ± 0.17	1.68 ± 0.18	1.75	>.05	Insignificant
5.	IVSd	0.93 ± 0.16	1.29 ± 0.26	4.94	<.001	Highly signi
6.	IVSs	1.57 ± 0.16	1.65 ± 0.19	1.37	>.05	Insignificant
7.	EF slope	91.2 ± 15.3	62.4 ± 15.8	5.69	<.001	Highly signi
8.	E	83.8 ± 4.5	69.1 ± 10.8	4.94	<.001	Highly signi
9.	A	52.8 ± 3.9	67.1 ± 10.1	5.17	<.001	Highly signi
10.	LVEF	70.2 ± 3.9	72.5 ± 11.8	0.72	>.05	Insignificant
11.	LV mass Index	95.7 ± 9.78	140.5 ± 31.2	5.74	<.001	Highly signi

PREVALENCE OF CARDIAC DYSFUNCTION IN HYPERTENSIVE PATIENTS WITH C.H.F

Related to Table V



Result

The difference in the mean value of LV wall thickness (end diastolic) between normotensive and hypertensive individuals was highly significant ($p < 0.001$) for LVPWd & IVSd whereas it was insignificant ($p > 0.05$) at end systole (Table VI).

The parameters of diastolic function showed high significant difference between control & study groups ($p<0.0001$) for EF slope, E & A.

Rest of the parameters are not different significantly the two groups.

Table.VII Spectrum of echocardiographic findings in patients with diastolic dysfunction only

S.No	Parameter	Case with only diastolic dysfunction *n = 19*	Percentage	Total cases with diastolic dysfunction *n = 22*	Percentage
1.	Reduced EF slope	19	100	22	100
2.	Altered E/A	19	100	22	100

Result

EF slope was reduced and E/A ratio altered in all the cases with diastolic dysfunction.

Table.VIII Comparison of echocardiographic parameters of LV diastolic function between control and study group.

S.No.	Parameter	Normotensive *(n=15)* Mean ± SD	Hypertensive with diastolic dysfunction *(n=22)* Mean ± SD	t	p	Inference
1.	EF slope	91.2 ± 15.3	54.5 ± 8.7	9.00	<.001	Highly signi
2.	E	83.8 ± 4.5	65.7 ± 6.8	9.77	<.001	Highly signi
3.	A	52.8 ± 3.9	71.9 ± 6.6	9.8	<.001	Highly signi

Result

The decrease in the value of EF slope and the increase in A is statistically highly significant in hypertensive subject leading to diastolic dysfunction.

Table.IX Spectrum of echocardiographic findings in patients with systolic dysfunction only

S.No.	Abnormal	Case with only Systolic dysfunction *(n=1)*	Percentage	Total cases of systolic dysfunction *(n=4)*	Percentage
1.	Reduced LVEF	1	100	4	100
2.	Increase LVId	1	100	4	100
3.	Increased LVIDs	1	100	4	100

Result

Increased LV dimension and reduced ejection fraction was present in all the cases of systolic dysfunction.

Table.X Comparison of echocardiographic parameters of systolic function between control and study group

S.No.	Parameter	Normotensive *(n=15)*	Hypertensive with systolic dysfunction *(n=4)*Mean ± SD	t	p	Inference
1.	LVEF	70.2 ± 3.9	50.75 ± 3.6	8.5	<.001	Highly signi
2.	LVIDd	4.59 ± 0.39	5.8 ± 0.18	5.7	<.001	Highly signi
3.	LVIDs	3.03 ± 0.57	4.35 ± 0.13	4.35	<.001	Highly signi

Result

The decrease in ejection function of LV and increase in its internal dimension is highly significant statistically in hypertensive patients leading to systolic dysfunction.

Table.XI Comparison of LVH detection by X-ray, ECG and echocardiography among 30 cases of hypertension

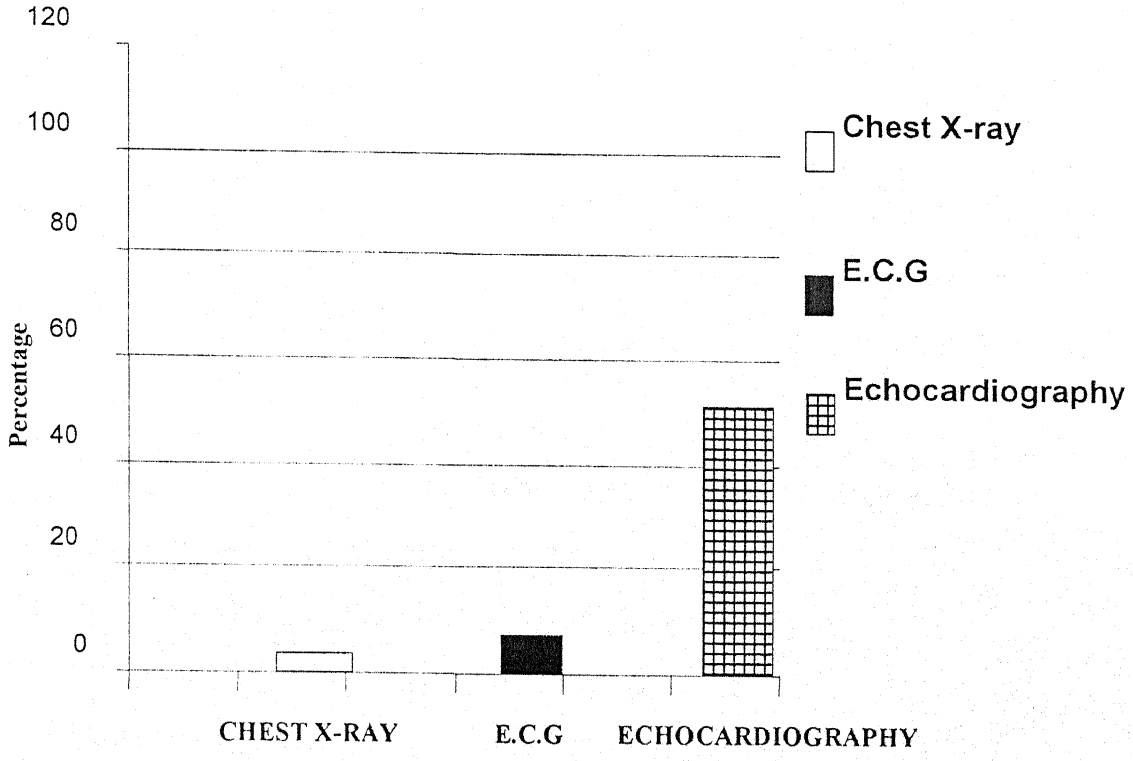
S.No.	Method	Hypertensive cases with LVH	Percentage
1.	Chest x-ray	1	3.33
2.	ECG	2	6.66
3.	Echocardiography	16	53.3

Result

By echocardiography, LVH was detected in 53.3% of hypertensives whereas ECG could detect it in only 6.66% and Chest radiography in 3.3% cases of hypertension.

COMPARISON OF L.V.H. DETECTION METHODS

Related to Table XI



In order to find out the relation of LV thickness with LV systolic and diastolic function the cases were further analyzed as follows.

Table.XII Relationship of LV functions with LVH in cases of hypertension

S.No.	Dysfunction	Total No. Of cases *n=30*	No. of cases with LVH *n=16*	Percentage
1.	Only diastolic	19	13	68.4
2.	Only systolic	1	1	100
3.	Both	3	2	66.6
4.	No	7	-	-
5.	Total diastolic	22	15	68.18
6.	Total systolic	4	3	75
7.	Both dysfunction with CHF	1	1	100
8.	Both dysfunction without CHF	2	1	50

Result

Prevalence of LVH hypertensive cases is as follows:-

In only diastolic dysfunction cases 68.4%.

In patients with systolic dysfunction 100%.

66.6% in patients with both dysfunction.

No LVH in hypertensive patients with normal LV function.

68.18% in total patients with diastolic dysfunction.

In 75% of total systolic dysfunction

Table.XIII Comparison of echocardiographic parameters of LV wall thickness between normotensive subjects and hypertensives

S.No.	Parameter	Normotensive Mean ± SD	Hypertensive Mean ± SD	t	p	Inference
1.	LVPWd	0.93 ± 0.17	1.3 ± 0.27	4.87	.001	Highly signi
2.	IVSd	0.93 ± 0.16	1.29 ± 0.26	4.94	.001	Highly signi

Result

The increase in the thickness of LV wall in hypertensive group was statistically highly significant (t = 4.87 & 4.94, p <.001).

Table.XIV Comparison of echocardiographic parameters of diastolic function between hypertensive patients with and without LVH

S.No.	Parameter	Diastolic dysfunction without LVH Mean ± SD	Diastolic dysfunction with LVH Mean ± SD	t	p	Inference
1.	EF slope	61.7 ± 6.4	52.9 ± 10.1	1.89	>.05	Insignificant
2.	E	68.8 ± 6.1	70.0 ± 8.8	20	>.05	Insignificant
3.	A	71.0 ± 6.7	72.7 ± 6.7	5	>.05	Insignificant

Result

The echocardiographic parameters of diastolic function did not show significant function between hypertensive patients with and without LVH.

Table.XV Correlation between LV dysfunction and duration of hypertension

S.No.	Duration In yrs.	No. of Hypert--ensive cases *n=30*	No. of Hypertensive cases with diastolic dysfunction *n=22*	Percentage	Hypertensive cases with systolic dysfunction *n=4*	Percentage
1.	< 5	14	7	50	2	14.28
2.	5 – 10	10	9	90	-	--
3.	> 10	6	6	100	2	33.3

Result

In hypertensive cases with less than five years duration of hypertension 50% had diastolic dysfunction and 14.28% had systolic dysfunction.

In cases with 5 – 10 years, none had systolic dysfunction whereas 90% had diastolic dysfunction.

In more than 10 years duration, all had diastolic dysfunction and 33.3% had systolic dysfunction.

Table.XVI LV dysfunction in hypertensive patients of different age groups

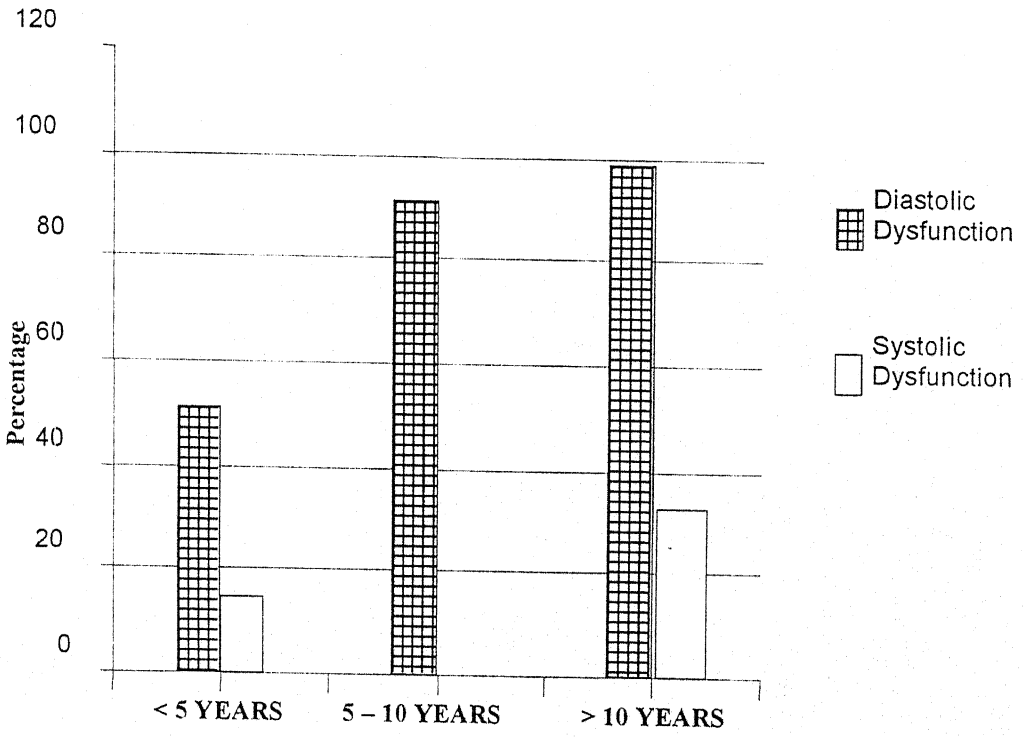
S.No.	Age groups	Hypertensive cases *n=30*	Hypertensive cases with diastolic dysfunction *n=22*	Percentage	Hypertensive cases with systolic dysfunction *n=4*	Percentage
1.	31 – 40 Yrs.	6	3	50	1	16.6
2.	41 – 50 Yrs.	10	7	70	-	-
3.	51 – 60 Yrs.	10	8	80	3	30
4.	61 – 70 Yrs.	4	4	100	-	-

Result

Systolic dysfunction was present in 16.6% in the 4^{th} decade 30% in 6^{th} decade. Diastolic dysfunction was present

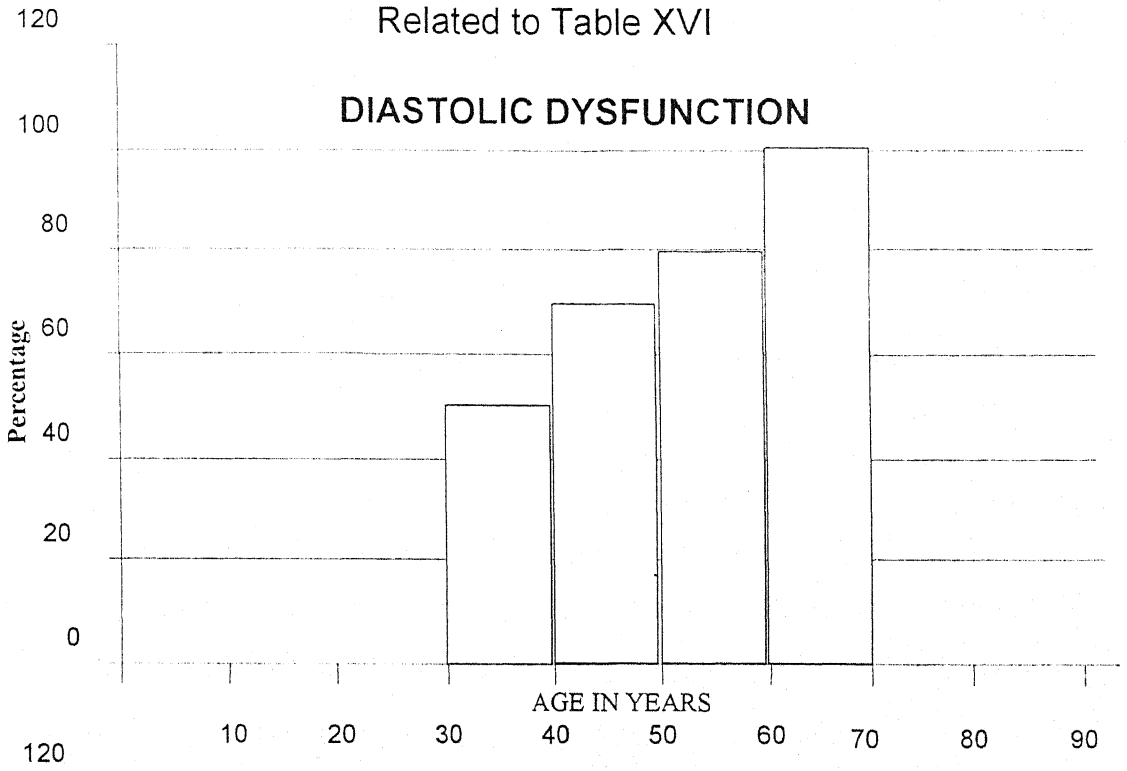
CORRELATION BETWEEN L.V DYSFUNCTION AND DURATION OF HYPERTENSION

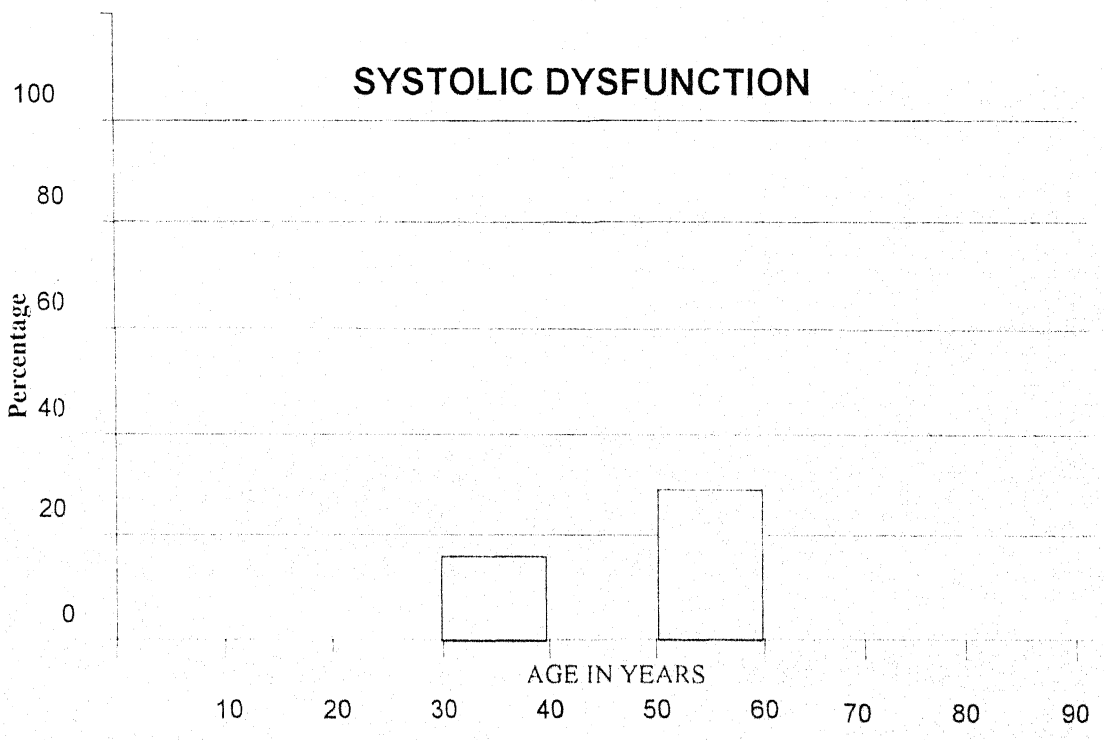
Related to Table XV



L.V DYSFUNCTION IN HYPERTENSIVE PATIENTS IN DIFFERENT AGE GROUPS

Related to Table XVI





in 50% in 4th decade and increased with age till 100% in 7th decade.

Table.XVII Relationship of risk factors with diastolic dysfunction in hypertensive subjects

S. No.	Clinical parameter	Without diastolic dysfunction Mean ± SD	With diastolic dysfunction Mean ± SD	t	p	Inference
1.	Age	44.6 ± 10.7	51.4 ± 9.7	1.59	>0.5	Insig.
2.	Duration	4.06 ± 4.47	6.86 ± 4.5	1.46	>0.5	Insig.

Result

The risk factor association of age of patients and duration of hypertension did not show statistically significant increase in cases with diastolic dysfunction when compared with those having normal diastolic function.

Table.XVIII LVH in hypertensive patients of different age groups

S. No.	Age Group	Hypertensive Cases *(n=30)*	Hypertensive cases with LVH *(n=16)*	Percentage
1.	31 – 40 years	6	2	33.3
2.	41 – 50 years	10	5	50
3.	51 – 60 years	10	5	50
4.	61 – 70 years	4	4	100

Result

33.3% cases had LVH among hypertensive in 4th decade. In the 5th and 6th decade LVH was relevant equally i.e. in 50% cases. All the patients had LVH in 7th decade.

Table.XIX Correlation between LVH and duration of hypertension

S. No.	Duration in years	Hypertensive Cases *(n=30)*	Hypertensive cases with LVH *(n=16)*	Percentage
1.	5	14	4	28.5
2.	5 – 10	10	6	60
3.	10	6	6	100

Result

LVH was present in 28.5% cases with hypertension of less than five years duration. It increased to 60% in cases with 5 – 10 years duration and all the cases has LVH in hypertension of more than 10 years.

Table.XX Relationship of risk factors with LVH in hypertensive individuals

S. No.	Clinical Parameter	without LVH Mean ± SD	with LVH Mean ± SD	t	p	Inference
1.	Age	45.8 ± 9.4	52.9 ± 10.2	1.9	.05	Not significant
2.	Duration	3.68 ± 3.05	8.25 ± 4.74	2.9	.01	Significant

Result

Age of cases of test group was not increased significantly in persons with LVH whereas the increase in duration of hypertension was statistically significant in person having LVH.

CORRELATION BETWEEN L.V.H AND DURATION OF HYPERTENSION

Related to Table XIX

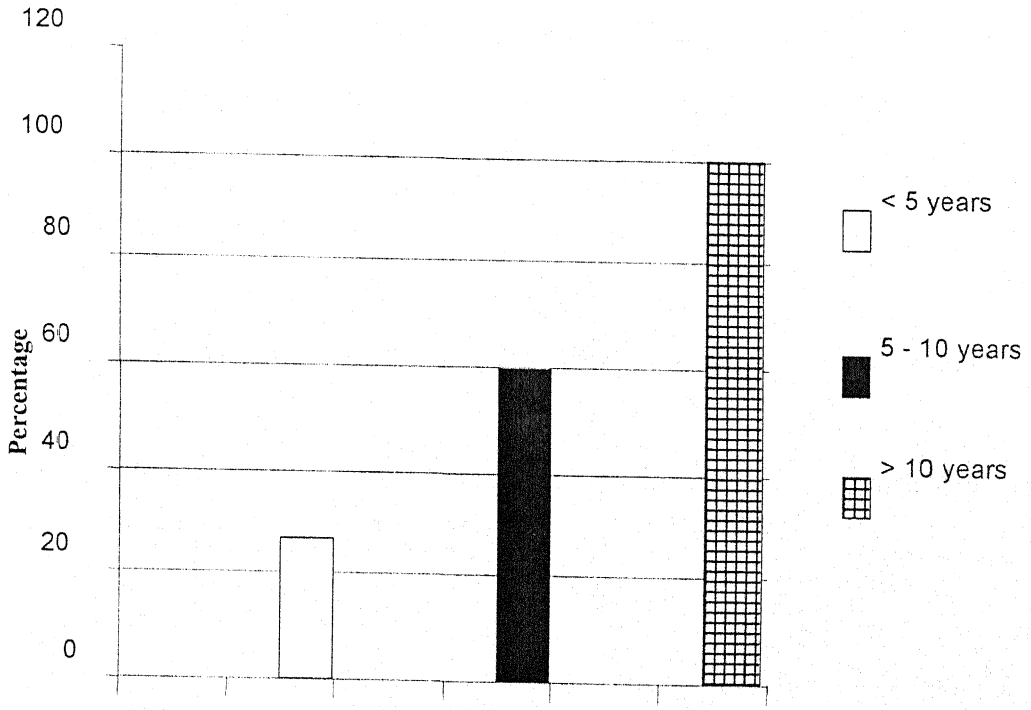


Table.XXI Correlation between severity of hypertension and cardiac dysfunction.

S No.	Severity of Hypertension	Hypertensive cases *(n=30)*	Diastolic dysfunction cases *(n=22)*	Percentage	Systolic dysfunction cases *(n=4)*	Percentage
1.	Stage I	12	9	75	1	8.3
2.	Stage II	10	7	70	-	-
3.	Stage III	8	6	75	3	37.5

Result

75% had diastolic dysfunction and only 8.3% had systolic dysfunction in persons with stage I hypertension. There was no systolic dysfunction in the stage II group only diastolic dysfunction in 70% cases. In patients with stage III hypertension 37.5% had systolic dysfunction and 75% had diastolic dysfunction.

Table.XXII Correlation between severity of hypertension and LVH

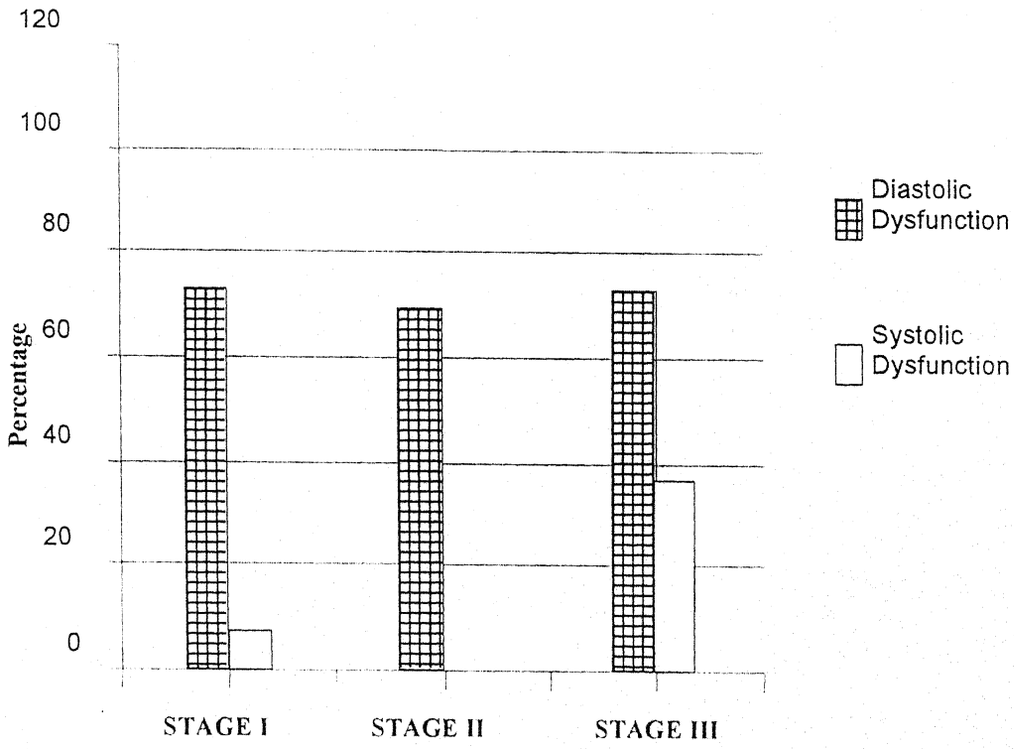
S No.	Severity of Hypertension	Hypertensive cases *(n=30)*	Cases with LVH *(n=26)*	Percentage
1.	Stage I	12	5	41.6
2.	Stage II	10	5	50
3.	Stage III	8	6	75

Result

In patients with stage I hypertension, LVH was present in 41.6%. This increased to 50% in stage II group and 75% in stage III hypertension.

CORRELATION BETWEEN SEVERITY OF HYPERTENSION AND L.V DYSFUNCTION

Related to Table XXI



CORRELATION BETWEEN SEVERITY OF HYPERTENSION AND L.V.H

Related to Table XXII

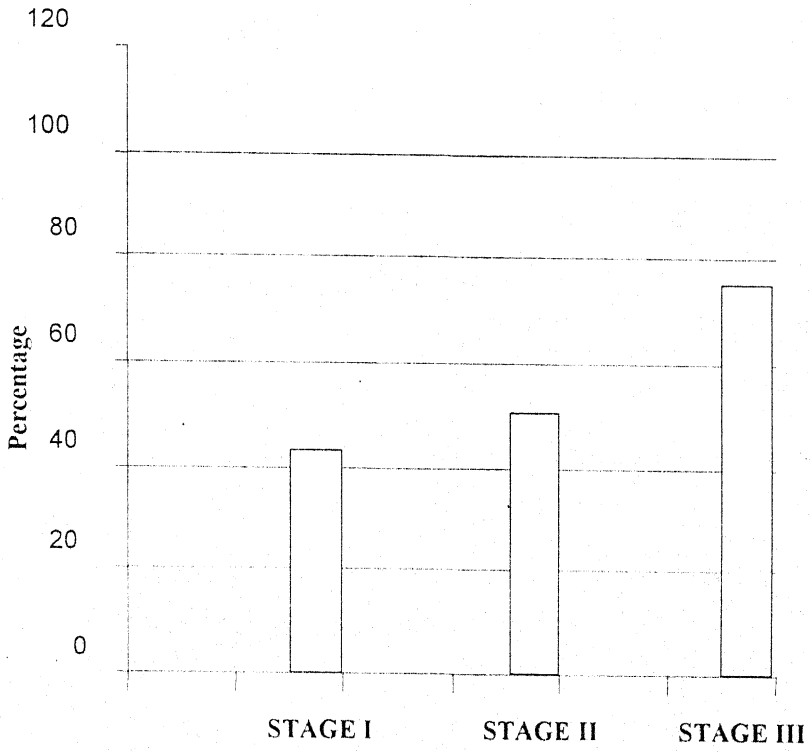


Table.XXIII Sex wise distribution of cases of LVH and LV dysfunction

S No.	Hypertensive cases	Male *(n=20)*	Percentage	Female *(n=10)*	Percentage
1.	LVH cases	10	50	6	60
2.	Cases with diastolic dysfunction	14	70	8	80
3.	Cases with systolic dysfunction	4	20	-	-

Result

Hypertensive females presented with LVH in 60% and diastolic dysfunction in 80%. Male had LVH in 50%, diastolic dysfunction in 70%, and systolic dysfunction in 20%.

Table.XXIV Correlation between LVH and LV dysfunction and control of blood pressure

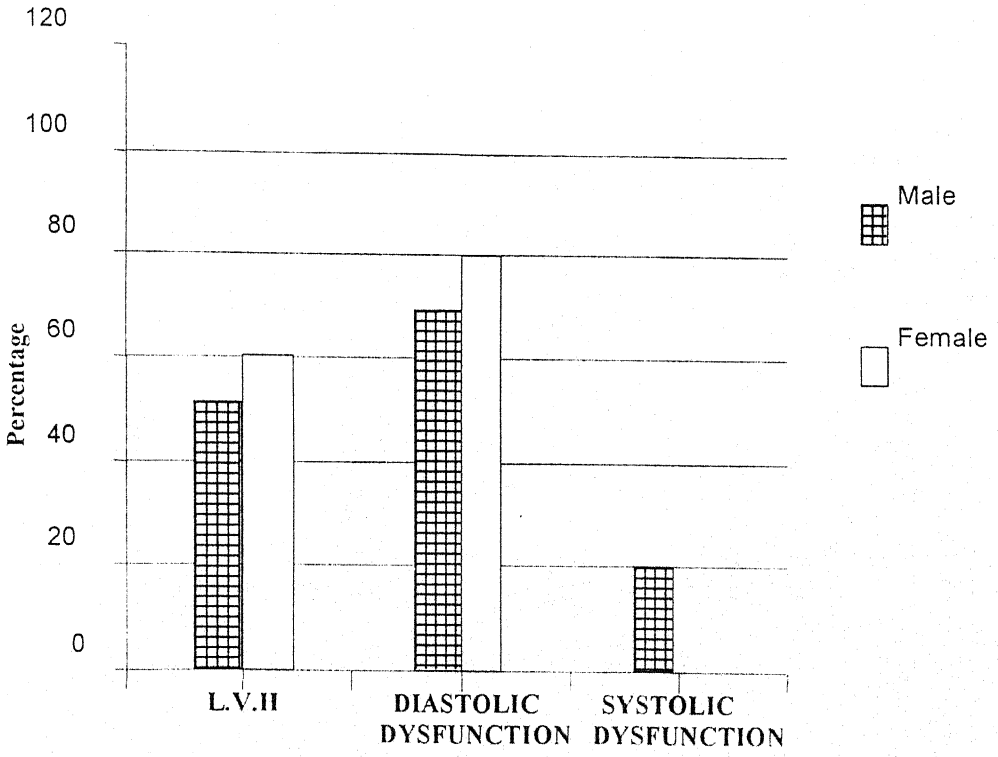
S No.	Cases Hypertensive Cases	B. P. control satisfactory *(n=16)*	Percentage	B.P. control unsatisfactory *(n=14)*	Percentage
1.	LVH cases	6	37.5	10	71.4
2.	Cases with diastolic dysfunction	11	68.75	11	78.5
3.	Cases with systolic dysfunction	1	6.25	3	21.42

Result

Hypertensives with satisfactory control of B.P. had LVH in 37.5%, diastolic dysfunction in 68.75% and systolic dysfunction in 6.25%. Those with unsatisfactory B.P. control had LVH in 71.4%, diastolic dysfunction in 78.5%, and systolic on 21.42%.

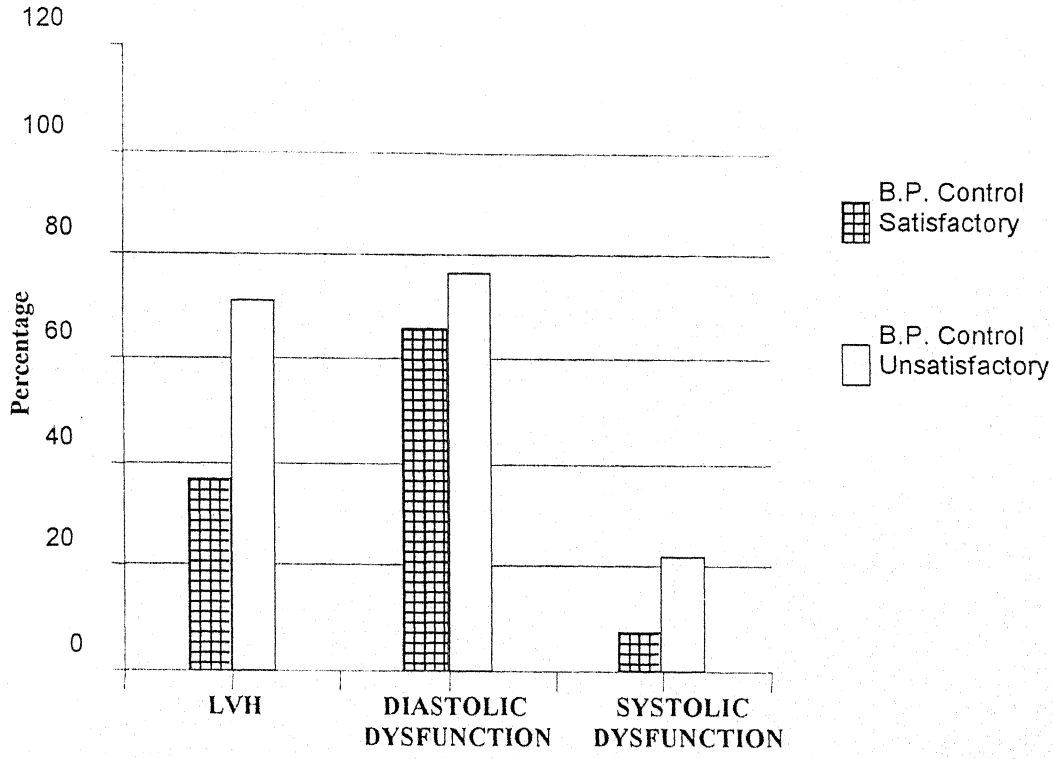
SEX – WISE DISTRIBUTION OF CASES OF L.V.H AND L.V DYSFUNCTION

Related to Table XXIII



CORRELATION OF L.V.H AND L.V DYSFUNCTION WITH CONTROL OF BLOOD PRESSURE

Related to Table XXIV



Discussion

DISCUSSION

The present study was undertaken to assess left ventricular systolic and diastolic functions by means of echocardiography (M–mode, 2D and Doppler) according to the criteria and conventions laid down by the American Society of Echocardiography, in 45 cases comprising of 15 normal subject and 30 cases of systemic hypertension.

Although conventional ECG and radiography provide some information about the cardiac anatomy, they remain silent regarding myocardial functional impairment. Contrast ventriculography carries risks of nephropathy, anaphylaxis and diuresis and may influence ventricular function.

More recently echocardiography has been the primary method for determining indices of LV functions.

Out of these we have chosen echocardiography as this can be employed safely in any setting without patient preparation, discomfort or inconvenience.

The age wise distribution of hypertensive cases in various age groups has been well highlighted by many workers like Janeway (1913), Bell and Clawson (1928), Bechgaard (1946) and Paulwood (1968). In our study, the age of the patients with systemic hypertension ranged from 31 – 68 years with mean of 49.3 ± 9.8. Maximum number of cases were between 41 – 60 years being equally divided in 5^{th} and 6^{th} decades.

The male female ratio of 2 : 1 in conformity with the study of Bell and Clawson (1928) and Gaur et al (1956). However, this could be due to more number of male patients attending our hospital then female.

DISCUSSION ON NORMAL SUBJECTS

For standardization the echocardiographic parameters in the normal healthy Indian controls as found in the present study are as follows:

Parameter	**Range**	**Mean ± SD**
LVIDd	4.2 – 5.4 cm	4.59 ± 0.39
LVIDs	2.2 – 4.0 cm	3.03 ± 0.57
LVPWd	0.7 – 1.2 cm	0.93 ± 0.17
LVPWs	1.3 – 1.8 cm	1.56 ± 0.17
IVSd	0.7 – 1.2 cm	0.93 ± 0.16
IVSs	1.1 – 1.8 cm	1.57 ± 0.16
EF slope	70 – 120	91.2 ± 15.3
E	80 – 90 m/s	83.8 ± 4.5
A	47 – 61 m/s	52.8 ± 3.9
LVEF	65 – 76 %	70.2 ± 3.9
LV mass index	81 – 98 gm/m^2	95.7 ± 9.78

Echocardiographic parameters for normal subjects as found by various workers are as follows:

LVIDd (cm)	**Range**
H. Feigenbaum (1994)	3.5 – 5.7
I Schnittger et al (1983)	3.5 – 6.0
Sam Kaddoura (2002)	3.5 – 5.6
Braunwald (2001)	3.5 – 5.7

LVIDs (cm)	Range
H. Feigenbaum (1994)	2.3 – 3.9
I Schnittger et al (1983)	2.1 – 4.0
Sam Kaddoura (2002)	2.0 – 4.0

IVS (cm)	Range
Sam Kaddoura (2002)	0.6 – 1.2 (diastolic) 0.9 – 1.8 (systolic)
Braunwald (2001)	0.6 – 1.1 (diastolic)
H. Feigenbaum (1994)	0.6 – 1.1 (diastolic)

LV Posterior Wall (cm)	Range
Sam Kaddoura (2002)	0.6–1.2 (diastolic) 0.9 – 1.8 (systolic)
Braunwald (2001)	0.6 – 1.1 (diastolic)
H Feigenbaum (1994)	0.6 – 1.1 (diastolic)

LVEF(%)	Range
Sam Kaddoura (2002)	50 – 85
H Feigenbaum (1994)	61± 3.4(range ± SD)
Harrison (15th ed.)	50 – 80 or 67 ± 8

E (m/s)	Range
Jae, k Oh, Seward (1994)	0.86 ± 0.16

A (m/s)	Range
Jae, K Oh, Seward (1994)	0.56 ± 0.13

EF slope	**Range**
Feigenbaun (1994)	90±16.5(Mean±SD)
Fujii et al (1979)	84±5.2 (Mean ± SD)

LV mass index (gm/m^2)	**Range**	
For normal Indian population		
Trivedi S.K (1991)	80.4±20.71 for ♂	
	72.9±28.18 for ♀	
Upper limit		
Trivedi S.K	122.06 in ♂	Indian
	110 in ♀	Indian
Ghanum Wisam et al (2000)	134 in ♂	Western
	110 in ♀	Western
Levy D et al (1988)	131 in ♂	Western
	100 in ♀	Western

As evident from the above values, the various parameters usually correspond with those of the present study.

In control subjects age & sex did not affect the various echocardiographic parameters used in assessing LV functions. Ciro et al (1984) and Dreslinski et al (1981) also found echocardiographic parameters uninfluenced by age; and till date the various works reported on the subject has not found any discrimination in echocardiographic parameters with respect to sex.

But Pearson et al (1987), have found decrease in EF slope and altered E/A ratio with increase in age. Increase in LV wall thickness has been reported by Savage et al. Probably the smaller size in higher age group in this study has resulted in this difference in Indian subjects.

Discussion on echocardiography vs. ECG and radiography for detecting LVH in systemic hypertension

Echocardiography showed LVH in 53.3% cases of hypertension as given in table No. XI. In these patients ECG indicated LVH in only 2 cases (6.6%), while radiography was even a poorer parameter which suggested LVH in only one case (3.3%). Echocardiography therefore, was found to be far superior method to detect earliest increase in the girth of LV wall. Early detection may be useful in predicting not only the prognosis but also highlighting therapeutic measure to control hypertension, which would have otherwise been treated as without target organ involvement.

In the study of Savage et al (1989), 3% cases were found to have LVH in hypertensives and 5% by chest x-ray while echocardiography detected it in 67%. Pearson, Gudipati et al (1987), found that out of 15 patients of hypertension, only one met ECG criteria of LVH (similar to present series) while 8 cases have echocardiographically proved LVH. Findings is also in conformity with Woythaler JN et al (1983) and Dougherty et al (1984), who found quantitative M-mode echocardiography more sensitive and specific as compared with electrocardiographic voltage, and Este's criteria as an index of hypertrophy.

Discussion on LVH and LV dysfunction occurring in systemic hypertension

In the present series LV dysfunction was found in 76.6% hypertensive subjects while it was normal in rest 23.3%. Savage et al (1989), found deranged LV function in 60% cases of hypertension. Many other workers have similarly found LV dysfunction in most cases of hypertension.

Among the hypertensive cases 73.3% had diastolic dysfunction while only 13.3% had abnormal systolic function. Combined dysfunction was found in only 10% cases. These findings are consistent with so many other workers who also found systolic dysfunction uncommon in such cases while diastolic dysfunction was the rule (Pearson and Labovitz, 1987). Only Savage et al (1989) have given contradictory results i.e. 6% diastolic dysfunction and 15% systolic dysfunction in hypertensive cases.

The incidence of LVH was 53.3% in this study by echocardiography, among cases of systemic hypertension. Similar incidence is reported by Savage et al (1989), who found it in 615 cases of hypertension by echocardiographic measurements. All the cases with LVH has LV dysfunction and that too mostly in the form of diastolic dysfunction (100%), while systolic or combined dysfunction occurred in only 18% cases of LVH. In patients with combined dysfunction if LVH was present, it was of eccentric type.

This also is in agreement with various authors. Smith et al (1985), Pearson & Labovitz (1987), and Papademetriou et al (1985), found abnormalities in diastolic properties of left

ventricle in patients with LVH secondary to systemic hypertension, an usual finding.

In this present study diastolic dysfunction was present in 6 cases without LVH and one case of systolic dysfunction was present without LVH among hypertensives. Inouye et al (1984) has also observed LV diastolic dysfunction without LVH. The occurrence of diastolic dysfunction preceding LVH could be explained on the basis, that factors beside increase in LV mass are also responsible for altered LV filling dynamics. The other possibility is that in such patients although increase in LV mass has occurred but yet this increase is not sufficient enough to meet the criteria for LVH by echocardiography.

Inouye et al (1985) also observed that severity of LV diastolic dysfunction increases in hypertensives with LVH. In the present study this correlation was not statistically significant ($p > .005$).

Discussion on echocardiographic parameters of LVH and LV systolic and diastolic dysfunction

Left ventricular posterior wall thickness at end diastole (LVIDd) and inter ventricular septal thickness at end diastole (IVSd) were considered to detect LVH. Various workers have used these parameters for the same purpose.

For assessing diastolic function, mitral EF slope, Peak velocity in rapid ventricular filling-E, peak velocity during atrial contraction-A, ratio of E/A, were considered in this study. For systolic function assessment, ejection fraction of left ventricle LVEF, Left ventricular internal dimension at end

diastole and end systole (LVIDs and LVIDd) were taken into account. LVEF is sensitive precursor for LV systolic deterioration in patients with hypertension (Qirko S et al 1999). The LVPWd and IVSd was1.3 cm ± 0.27 and 1.29 cm ± 0.26 respectively, in this study in hypertensives and, the increase in the value as compared to normal subjects was statistically highly significant (t=4.87, p < 0.001;t=4.94, p 0<.001) respectively. Savage et al (1989), similarly found highly significant results (p < .001). Safar et al (1990), reported 1.11 cm ± .03 and 1.12 cm ± .04 for LVPWd and IVSd, and p < 0.01 and p < 0.001 respectively in cases of sustained hypertension. This closely matches our findings, LVPWd was found to be 1.5 cm ± 0.1 by Grossman et al (1990), 1.4 ± 0.4 by Ciro et al (1984) in cases of chronic pressure overload. These slightly increased values obtained by these authors could be due to the fact that they included few cases of aortic stenosis also in their study.

In the present series EF slope, E and A in hypertensives, as parameters for diastolic dysfunction, were significantly altered compared to normal subjects (t = 5.69, p < .001; t = 4.94, p < 0.001; t = 5.17, p < 0.001). The mean of EF slope , E and A was 62.4 ± 15.8, 69.1 m/s ± 10.8 and 67.1 m/s ± 10.1 respectively. This is in agreement with most of the authors. (Pearson et al (1987), Savage et al (1989), found EF slope decrease, statistically significant (p < 0.01) in hypertensives with diastolic dysfunction.

The decrease in LVEF was not significant (p < 0.05) in our study statistically because only 4 out of 30 cases of hypertension had decreased EF, which is consistent with the

fact that systolic dysfunction is uncommon in systemic hypertension. Besides, some patients of hypertension with no dysfunction in the early stage, and with even diastolic dysfunction showed supranormal systolic function by increase in LVEF. Thus the average of LVEF in hypertensives was not decreased significantly. The mean EF in moderate hypertensives as found by Fujii et al (1994), 68% ± 10 is close to the value obtained in present series i.e. 72.5 ± 11.8. However Savage et al (1989), found EF reduction in hypertensives statistically significant.

There was statistically a highly significant alteration in the mean values of LVEF, LVIDd, LVIDs, in hypertensive patients with systolic dysfunction, which is in agreement with Dougherty et al (1984) and others.

In the present study LVIDd and LVIDs was not increased significantly in hypertensives as per statistics ($p > .05$). Their mean values were 4.8 cm ± 0.52 and 3.4 cm ± 0.67. This is in conformity with the results of following workers: Ciro et al (1984), found LVIDd 4.6 ± 0.09 cm; Safar et al (1990), reported LVID 4.63 cm ± 0.1 and LVIDs 3.47 cm ± 0.11. Only the findings of Savage et al (1989), is different in this regard who obtained significant reduction in LVEF in hypertensives ($p < 0.05$).

Discussion on risk factor correlation with LV dysfunction in cases of systemic hypertension

In the present study LV dysfunction correlated well with advancing age of the patient. This is in agreement with the

study of Savage et al (1989) who also found decrease in EF with increase in age.

No definite correlation was found between severity of hypertension and LV dysfunction in this which is consistent with conclusions of Savage et al (1989).

LVH in hypertensive patients correlated positively with increase in age, during and severity in the present series. Savage et al (1989), also found positive correlation with age of hypertensive and severity and duration of hypertension.

In this study there was positive correlation of LV dysfunction with increase in age and satisfactory control of blood pressure. LV dysfunction was present in asymptomatic cases of hypertension also which is important from therapeutic point of view, for otherwise these cases would have been treated as without target organ damage. No sex wise preponderance of LV dysfunction was found as in Inouye et al 1985 & others.

Discussion on patients with CHF consequent to systemic hypertension

Congestive heart failure is a common and often lethal complication of chronic systemic hypertension. In the present series 3 cases of systemic hypertension also had CHF clinically. On echocardiographic evaluation, diastolic dysfunction, systolic dysfunction and combined dysfunction, was found to be equally prevalent in them i.e. 33%.

Earlier studies implicated that in CHF, only systolic functions of LV was deranged, but now CHF cases with isolated diastolic or combined dysfunction has been

recognized by Pearson and Labovitz (1987), Topol et al (1990). Among such CHF cases with diastolic dysfunction, systemic hypertension has been found to be a major cause.

Inouye et al (1984), postulated that systolic dysfunction is uncommon in the patients with stage I and stage II hypertension, even though this group forms the substrate from which many persons with CHF will emerge. In contrast, diastolic dysfunction is the rule. This is consistent with the early appearance of left atrial enlargement in the evolution on LVH and may explain the dyspnoea and pulmonary congestion, which are sometimes seen in hypertensive patients.

Our findings resemble those of Dougherty et al (1984), who also studied 188 patients with CHF : 64% of them had reduced LVEF while 36% had normal EF. Of the patients with normal EF, 65% were due to systemic hypertension.

This finding points out that such patients need little inotropic support, rather therapy should be aimed at improving the diastolic filling.

Echocardiography thus permits rapid and noninvasive detection of cardiovascular complications in systemic hypertension, some of which would otherwise remain unrecognized or even unsuspected.

Conclusion

&

Summary

CONCLUSION AND SUMMARY

The following conclusion emerge from the present study:-

1. The age of systemic hypertension ranges from 31 - 68 years with mean of 49.3 ± 9.8.
2. Maximum number of cases fall between 41 and 60 years, being equally divided in 5^{th} and 6^{th} decades.
3. Male – Female ratio is 2 :1 in hypertensive individuals.
4. LV dysfunction is present in 76.6% of hypertensive subjects while, it remains normal in the remaining 23.3%. Left ventricular dysfunction may be either systolic or diastolic or both in such patients.
5. 73.3% cases of systemic hypertension have LV diastolic dysfunction including 63.3% cases of isolated diastolic dysfunction and 10% cases of combined systolic and diastolic dysfunction
6. L.V. systolic dysfunction occurs in 13.3% cases of systemic hypertension including 3.3% cases of isolated systolic dysfunction and 10% cases of combined L.V. systolic and diastolic dysfunction.
7. Of the cases of combined L.V. systolic and diastolic dysfunction, 3.3% cases present with CHF whereas 6.6% have no evidence of CHF.
8. CHF complicate hypertension in 10% cases.
9. In patients of CHF complicating hypertension, isolated LV

systolic dysfunction, isolated diastolic dysfunction and combined LV diastolic and systolic dysfunction is present in equal number of cases (33% in each group).

10. Echocardiographic parameters in cases of systemic hypertension with diastolic dysfunction are : Reduced E.F. slope and altered E/A ratio in all the cases (100%)
11. On comparing echocardiographic parameter of diastolic function in control and study group, the decrease in the EF slope and E value, and the increase in A, is statistically highly significant ($p < .001$, $t=9$; $p < .001$, $t=9.77$; $p < .001$, $t=9.8$ respectively).
12. Echocardiographic parameters in cases of systemic hypertension with systolic dysfunction are : Reduced LVEF and increased LVID in all the cases (100%).
13. On comparison of echocardiographic parameters of systolic function between control and study groups, the decrease in ejection fraction, and the increase in LVIDd and LVIDs, is statistically highly significant and all the parameters should be taken into consideration for evaluation of systolic function ($p < .001$, $t=8.5$; $p < .001$, $t=11.2$, $p < .001$, $t= 5.56$; $p < .001$, $t=5.52$; $p < .001$, $t=5.7$; $p < .001$, $t=4.35$).
14. (a) Echocardiographic evidence of LVH occurs in 53.3% cases of systemic hypertension. (b) LVH occurs in 68.4% cases of systemic hypertension presenting with diastolic dysfunction. (c) Isolated systolic dysfunction is also associated with LVH; (d) All the cases with LVH show LV dysfunction either systolic or diastolic or combined. (e) Diastolic dysfunction occurs in all the cases (100%) of

systemic hypertension with LVH. (f) Systolic dysfunction also occurs in all cases of systemic hypertension with LVH. (g) Both systolic and diastolic dysfunction can occur without LVH.

15. On comparison of echocardiographic parameter of LV wall thickness between control and hypertensive persons, the increase in LVPWd (1.3 ± 0.27) and IVSd (1.29 ± 0.26) in study group is statistically highly significant ($p < .001$, $t = 4.87$; $p < .001$, $t = 4.94$).

16. On apparent perusal, there is an increase in the incidence of LV diastolic dysfunction with increasing duration of systemic hypertension, but statistically, the increase in duration is not significant. There is no correlation between increase in duration of hypertension and systolic dysfunction.

17. LV diastolic dysfunction is also usually related to the age of the patient, as this increases with advancing age irrespective of the duration of hypertension; but systolic dysfunction has no correlation.

18. Incidence of LVH usually increases with advancing age in hypertensive cases being 33.3% in 4^{th} decade; 50% in 5^{th} and 6^{th} decades and 100% in 7^{th} decade.

19. Duration of hypertension is proportional to LVH and is found to be higher with increase in duration. The increase in duration in patients with LVH is statistically significant ($p < .01$, $t=2.9$).

20. There is no definite correlation between severity of hypertension and cardiac dysfunction although, maximum

number of cases of systolic dysfunction occurs in patients with stage III hypertension.

21. Severity of hypertension is related to LV wall thickness as the incidence of the latter increase with severity of hypertension being, 41.6% in stage I cases, 50% in stage II and 75% in stage III cases.

22. LVH and diastolic dysfunction is slightly more in female hypertensives whereas systolic dysfunction is present in males exclusively in the present series.

23. Hypertensive patients with satisfactory control of blood pressure have lesser complications like LVH and LV dysfunction, compared to those with poor control.

24. LV diastolic and systolic dysfunction is equally prevalent between symptomatic and asymptomatic groups of hypertensives.

25. LVH could be detected in 3.3% and 6.6% cases by chest radiograph and ECG respectively, while echocardiography could detect it in 53.3% cases of hypertension. Thus echocardiography is far superior laboratory tool for LVH detection compared to convention X-ray and ECG.

26. Assessment of systolic and diastolic dysfunction in cases of CHF complicating systemic hypertension has great clinical importance. In these patients because medications such as digitalis, vasodilators and diuretics, which are commonly employed to treat CHF, suspecting only systolic dysfunction, may in fact have an untoward effect on the patients, with coexistent or isolated diastolic dysfunction. While medications with known negative

inotropic effects such as calcium channel blockers and β blockers may prove beneficial.

27. Echocardiography provides a reliable, noninvasive and sensitive method for detecting accurately LVH and LV systolic and diastolic dysfunction, which have important diagnostic, therapeutic and prognostic implications in patients of systemic hypertension.

Appendix

APPENDIX

A	Mitral peak flow velocity during atrial contraction
CHF	Congestive heart failure
E	Mitral peak flow velocity during rapid filling phase
LVEF	Ejection fraction of left ventricle
IVSd	Inter ventricular septal thickness at end diastole
IVSs	Inter ventricular septal thickness at end systole
LVIDd	Left ventricular Internal dimension at end diastole
LVIDs	Left ventricular Internal dimension at end systole
LVH	Left ventricular hypertrophy
LVPWd	Left ventricular posterior wall thickness at end diastole
LVPWs	Left ventricular posterior wall thickness at end Systole

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